


Original Research Article

# Study on analysis of socio-demographic aspects, clinical profile, ECG changes, electrolyte status and outcome with acute yellow oleander seed poison patients in GDMCH, Dharmapuri

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## Abstract

**Introduction:** Ingestion of oleander seeds results in a clinical picture similar to that of digoxin overdose. Severely poisoned patients may die in dc shock resistant ventricular fibrillation. It can lead to severe Hyperkalaemia is a marker of a poor outcome in cardiac glycoside poisoning, which may worsen further toxicity and lead to serious Arrhythmias.

**Aim:** To analyse Clinical Profile, ECG Changes, Electrolyte Status among the patients with acute yellow Oleander seed Poisoning.

**Materials and methods:** All patients admitted with consumption yellow oleander poison within 48 hours was studied. The study was carried out over a nine-month-period beginning June 2016. Clinical parameters, CBC, Blood sugars, Serum Urea, Creatinine, Serum Na<sup>+</sup>, Serum K<sup>+</sup>, LFT, ABG, ECG, and Gastric aspirate analysis were done to analysis the changes on hourly basis.

**Results:** Among the 72 patients, the significant ECG abnormalities were found in 45(62.5%), 37.5% had normal rate and rhythm. No significant abnormalities were found in Renal and Liver functions. No significant change in Serum Sodium level was noted. But significant change in the Serum Potassium levels was noted in 28.1% of patients. In this 19.7% had Hyper-Kalemia and 8.4% had Hypo-Kalemia.

**Conclusion:** Oleander seed is still used as a suicidal agent. Oleander plant is easily available as an ornamental plant in urban, semi-urban and rural areas. ECG abnormalities were found in majority of the individuals. Prognosis was poor among those who presenting with Hypo-tension, Electrolyte disturbances especially those with Hyperkalaemia, and complex Arrhythmias.

## Key words

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Oleander seed, Hyperkalaemia, ECG abnormalities, Cardiac glycoside poisoning.

## Introduction

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The yellow oleander (*Thevetia Peruviana*) is an ornamental tree, which is common throughout the tropics and subtropics. It contains cardiac glycosides that are toxic to cardiac muscle and the autonomic nervous system [1]. Deliberate ingestion of oleander seeds has become a popular method of self-harm in India. Oleander is an ornamental tree of the Apocyanaceae family that is common throughout the tropics and subtropics. It is wide spread in India, Nepal and Srilanka [2]. Its flowers are used as offerings in the temple. Its sap contains cardiac glycosides (thevetins A and B and neriifolin) including the roots and the smoke produced from burning, toxic to cardiac muscle and the ingestion of its seeds results in a clinical picture similar to that of digoxin overdose [3-5]. Oleander leaf also contains other biologically active constituents that have antimitotic and insecticidal properties [4]. Oleander is also reported to have emetogenic, cathartic, insecticide, parasiticidic, anthelmintic, menstrual stimulant, and abortifacient activities. The majority of deaths occurring after ingestion of plant are due to yellow Oleander or 'pila kaner' (*cerebra thevetia*), pink eyed cerebra or 'sea mango' (*cerebra manghas*), and white oleander or 'kaner' (*nerium odorum*) are reported in South India. Poisoning with another related plant *cerebra odollum* is a common occurrence in Kerala with as many as 50% of the plant poisoning caused by this plant [5]. Adults have died after consuming oleander leaves in herbal teas.8 Accidental poisonings occur throughout the tropics particularly in children [6]. Many patients with moderate poisoning show PR interval prolongation and progression to atrioventricular (AV) dissociation. Severely poisoned patients may die in dc shock resistant

ventricular fibrillation. However, deliberate ingestion of yellow oleander seeds has recently become a popular method of self-harm. So, it was decided to study the yellow oleander poisoning in our place regarding the clinical profile, ECG changes and Electrolytes changes which decides the mortality and modification of which would improve the prognosis in our patients [7].

## Materials and methods

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All patients admitted with consumption yellow oleander poison within 48 hours was studied. The study was carried out over a nine-month-period beginning June 2016. A detailed history was obtained and patients were subjected to through clinical examination as well as to ECG soon after admission. They were assessed on an hourly basis for the first 6 hours, 12 hourly for next 72 hours then daily until complete recovery. Results of biochemical investigations including serum electrolytes will be entered in a pre-designed proforma. Serial ECGs was collected.

### Inclusion criteria

Patients admitted to the toxicology ward within 48 hours of ingestion of poison.

### Exclusion criteria

- Patients with history of cardiovascular disease.
- Patients on cardiac drugs.
- Patients with chronic renal failure.
- Patients on diuretics.

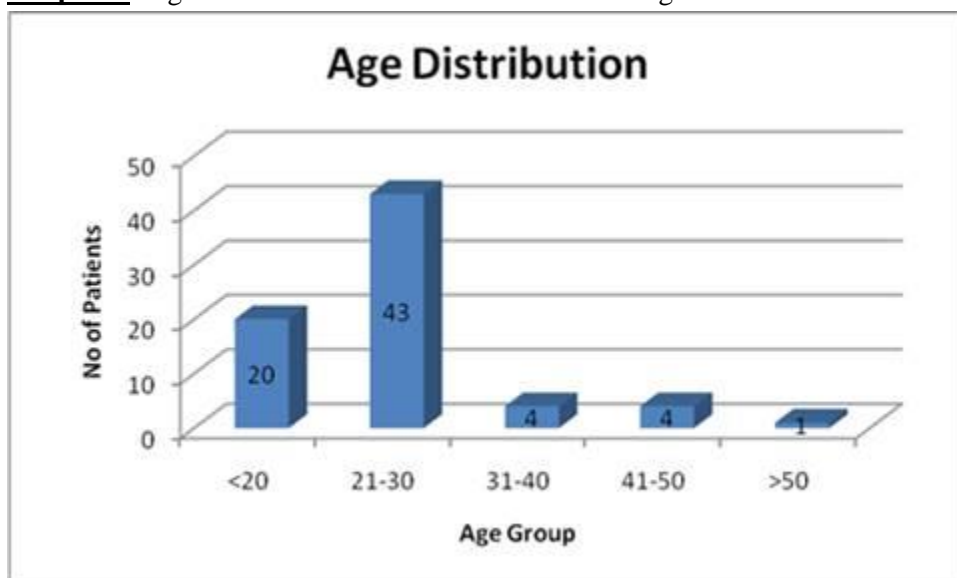
## Results

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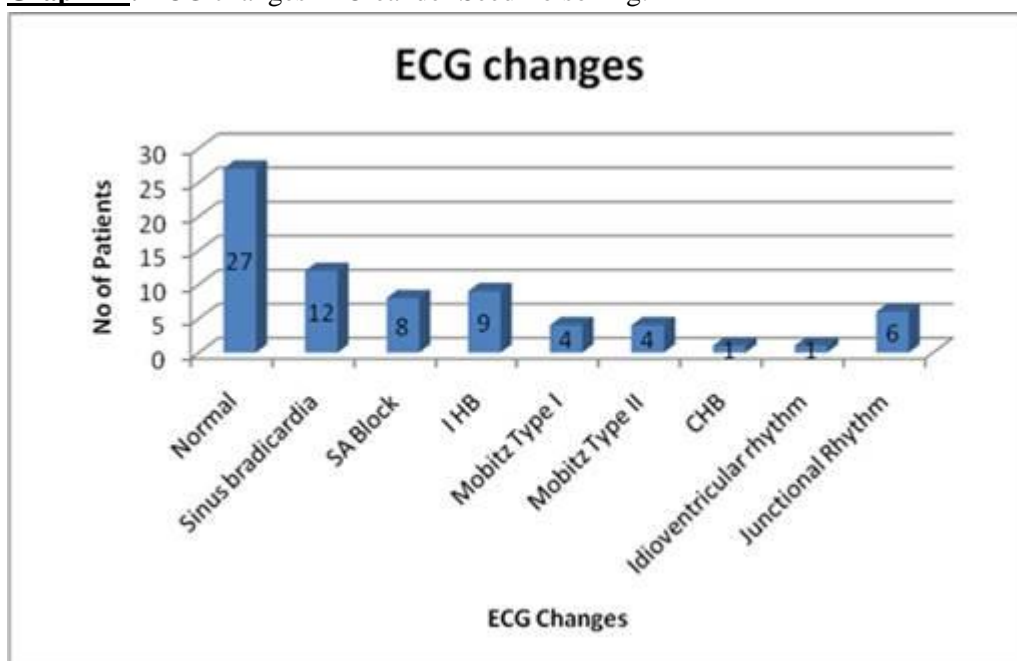
Age distribution of Oleander Seed Poisoning was as per **Graph – 1**. ECG changes in Oleander Seed Poisoning were as per **Graph – 2**.

Biochemical value levels in Oleander Seed Poisoning were as per **Table – 1**. Liver function test changes in Oleander Seed Poisoning were as per **Table – 2**.

**Graph - 1:** Age distribution of Oleander Seed Poisoning.



**Graph - 2:** ECG changes in Oleander Seed Poisoning.



**Table – 1:** Biochemical value levels in Oleander Seed Poisoning.

Sino	Values	Mean	S.D(+/-)
1.	Blood Sugar mg/dl	115.94	70.82
2.	Urea mg/dl	24.97	9.15
3.	Creatinine	0.85	6.18
4.	Sodium	125.54	8.20

**Table – 2:** Liver function test changes in Oleander Seed Poisoning.

Sl. No	Values	Mean	S.D(+/-)
1.	Bilirubin	0.72	0.10
2.	AST	24.24	14.32
3.	ALT	22.4	10.0
4.	Total Protein g/dl	6.5	0.50

## Discussion

In this series of 72 cases, the number of the patients between the age group of below 20 were 19 (26%), between 21-30 were 42 (58%) and remaining were 11 (16%). This young age group affected by exposure from the viable entity of any population both in terms of procurement and productivity. This case study and the case reports mentioned above throw light on the target age group for educative and preventive programs to reduce the incidence of oleander seed poisoning [8]. In GDMCH, females were exposed slightly more than male's population (51.4% versus 48.6%). Overall mortality in this study was 5 (5.5%). In age group of below 20 the mortality rate was 1 (1.4%), between 20-30 was 2 (2.7%) and 31-40 was 1 (1.4%) [9]. Among the 72 patients, the significant ECG abnormalities were found in 45(62.5%), 37.5% had normal rate and rhythm. In these 16.6% had Sinus Bradycardia, 12.5% and 11% respectively had II Degree Heart Block and I. Among the 11% of the II Degree Heart Block 5.5% with Mobitz Type II Block. 1.3% had the complete Heart Block. 11.11% had SA block and 8.3% presented with Junctional rhythm. 1.3% had idioventricular rhythm and in our study, Ventricular ectopics and Bundle branch blocks were not found. In Srilanka by Eddleston et al was observed among 89 seriously ill patients, 53% had AV node conduction block, 62% had sinus node block; 30% had conduction block affecting both nodes, 1% had ventricular tachycardias and 8% had ventricular ectopics [10]. In Thanjavur by Bobby et al, observed that 27 patients who had ECG abnormalities among the 51 patients, Sinus Bradycardia (59.25%), Sinus tachycardia (11.11%), T-wave inversion (7.4%), S-T depression(11.11%), SA Block (7.4%), I Degree

Block (11.11%), II Degree Block(3.7%), and Ventricular Tachycardia(3.7%). No significant abnormalities were found in Renal and Liver functions. Eddleston et al in Srilanka also noted similar observation. No significant change in Serum Sodium level was noted [11]. But significant change in the Serum Potassium levels was noted in 28.1% of patients. In this 19.7% had Hyper-Kalemia and 8.4% had Hypo-Kalemia. Among the 4 patients died in our series, 3 had Hyper-Kalemia [12]. Remaining one patient went to cardiac arrest immediately after admission; [13] so Laboratory investigation could not be done. Similarly studies conducted in Ananthapura in Silence by Eddleston et al revealed that the ECG changes and the mortality were higher in those who had Hyper-Kalemia. But exact incidence was not given [14].

## Conclusion

Oleander plants are used as ornamental plants throughout India. We have to avoid this. As conflict exists between the cardiologist and emergency care physician with reference to the indication for temporary pacemaker in the management of arrhythmia, there is a need for standard guidelines.

## Strengths and Limitations

The limitations were single centre study and children's are not included. Serum cardiac Glycosides (Digoxin) levels are not tested for the patients. Anti-Digoxin-AB treatment was not given [15].

## References

1. Pearn J. Oleander poisoning. In: Covacevich J, Davine P, Pearn J, eds.

- Toxic plants and animals: a guide for Australia, 2nd ed. Brisbane: William Brooks, 1989. P. 37-50.
- Langford SD, Boor PJ. Oleander toxicity: An examination of The human and animal toxic exposures. Toxicology, 1996; 109:1-13.
  - Sreeharan N, Putharasingam S, Ranjadayalan K, et al. Yellow oleander poisoning—clinical manifestations and prognostic criteria. JaVna Med J., 1985; 20: 100-1.
  - Saravanapavananthan N, Ganeshamoorthy J. Yellow Oleander poisoning - a study of 170 cases. Forensic Sci Int., 1988; 36: 247-50.
  - Fisch C, Surawicz B, eds. Digitalis. New York: Grune and Stratton, 1969.
  - Shaw D, Pearn J. Oleander poisoning. Med J Aust., 1979; 2: 267-9.
  - Brewster D. Herbal poisoning: a case report of a fatal yellow Oleander poisoning from the Solomon Islands. Ann Trop Pediatr., 1986; 6: 289-91.
  - Haynes BE, Bessen HA, Wightman WD. Oleander tea: Herbal draught of death. Ann Emerg Med., 1985; 14: 350-3.
  - Eddleston M, Ariaratnam CA, Meyer PW, et al. Epidemic of Self-poisoning with seeds of the yellow oleander tree (Thevetia peruviana) in northern Sri Lanka. Trop Med Int Health, 1999; 4: 266-73.
  - Eddleston M, Sheri V MHR, Hawton K. Deliberate Self-harm in Sri Lanka: an overlooked tragedy in the developing world. BMJ, 1998; 317: 133-5.
  - Dr. K.S. Narayana Reddy, The Essentials of Forensic Medicine and Toxicology; Twenty-fifth Edition, 2006.
  - Nathanson JA. Caffeine and related methylxanthines: Possible naturally occurring pesticides. Science, 1984; 226: 184-7.
  - Heywood VH, ed. Flowering plants of the world. Oxford: Oxford University Press, 1978.
  - Siddharth N. Shah, M. Paul Anand, API Textbook of Medicine, 8<sup>th</sup> Edition.
  - Ansford AJ, Morris H. Fatal oleander poisoning. Med J Aust., 1981; 1: 360-361.